

AMNIOTIC FLUID EMBOLISM (AFE)



What's the meaning of AFE

- Amniotic Fluid Embolism is a complex condition characterized by the abrupt onset of pulmonary embolism, shock and DIC, which is due to the entering of amniotic fluid into the maternal circulation.



AMNIOTIC FLUID EMBOLISM

- Overall incidence ranges from 1 in 8,000 to 1 in 80,000 pregnancies.
- 10% of maternal deaths in USA & 16% in U.K.
- 75 % of survivors are expected to have long-term neurologic deficits.
- If the fetus is alive at the time of the event, nearly 70 % will survive the delivery but 50% of the survived neonates will incur neurologic damage.



AMNIOTIC FLUID EMBOLISM

- **Race**

No racial or ethnic predilection exists.

- **Sex**

AFE only occurs in women.

- **Age**

Previously, advanced maternal age was believed to be a risk factor.

No relationship to age has been found in the National Amniotic Fluid Embolus Registry.



AMNIOTIC FLUID EMBOLISM

- Time of event:
 - During labor.
 - During C/S.
 - After normal vaginal delivery.
 - During second trimester TOP.
 - AFE syndrome has been reported to occur as late as 48 hours following delivery.



Etiology

- Pressure increasing:
 - uterine hypertonus
 - tetanic
 - oxytocin
- Open vessels: traumatic, laceration
- Membrane changing: fetal death,
 - dystocia
- Amniotic fluid itself
- Differ constitutions: Allergic reaction



Risk factors of AFE

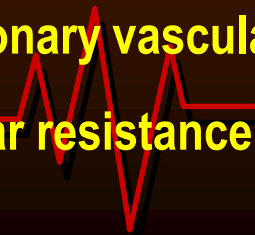
- Advanced maternal age
- Multiparity
- Meconium
- Cervical laceration
- Intrauterine fetal death
- Very strong frequent or uterine tetanic contractions
- Sudden fetal expulsion
- Placenta accreta
- Polyhydramnios
- Uterine rupture
- Maternal history of allergy or atopy
- Chorioamnionitis
- Macrosomia
- Male fetal sex
- Oxytocin (controversial)

**Nevertheless, these and other frequently cited risk factors are not consistently observed and at the present time
*Experts agree that this condition is not preventable.***

AFE

The cardiorespiratory effects of acute intravascular injection of amniotic fluid have been studied in pregnant ewes :

- The initial response was **hypotension**.
- A 40 % decrease in mean arterial pressure was followed by a 100 % **increase in mean pulmonary artery pressure**.
- Little change occurred in the left atrial pressure or the pulmonary artery wedge pressure.
- A 40 percent fall in cardiac output was associated with the rapid rise in pulmonary artery pressure.
- These changes resulted in a two- to threefold **increase in pulmonary vascular resistance** and a two- to threefold **decrease in systemic vascular resistance**.



Pathophysiology

- Poorly understood.
- Cotton (1996), has proposed a **biphasic model**.

Phase 1:

*Amniotic fluid and fetal cells enter the maternal circulation ⇒ biochemical mediators ⇒ pulmonary artery vasospasm ⇒ pulmonary hypertension ⇒ elevated right ventricular pressure ⇒ hypoxia ⇒ myocardial and pulmonary capillary damage, ⇒ left **heart failure** ⇒ **acute respiratory distress syndrome***

Phase 2:

*⇒ biochemical mediators ⇒ **DIC** ⇒ Hemorrhagic phase characterized by massive hemorrhage and uterine atony.*

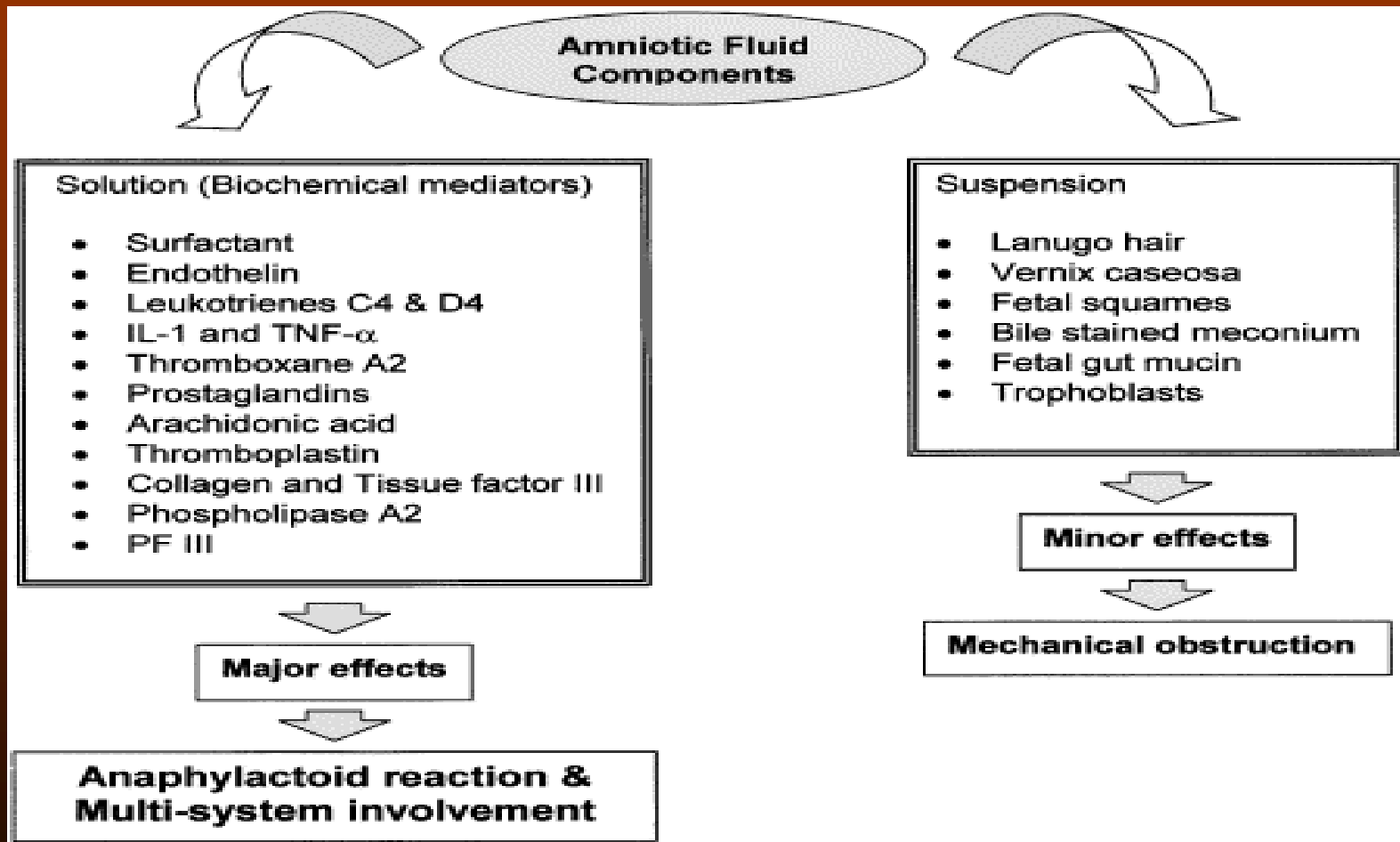


Pathophysiology

- The similar hemodynamic derangements seen with AFE syndrome , anaphylactic, and septic shock have led investigators to postulate a substance in amniotic fluid resulting in the release of primary and secondary endogenous mediators (i.e. arachidonic acid metabolites) which might also be responsible for the associated **coagulopathy** in AFE.
- The prevention of fatal hemodynamic collapse in experimental AFE with inhibitors of leukotriene synthesis would support an **anaphylactic mechanism** for AFE.



Pathophysiology



Clinical presentation

The classic clinical presentation of the syndrome has been described by five signs that often occur in the following sequence:

- (1) Respiratory distress
- (2) Cyanosis
- (3) Cardiovascular collapse **cardiogenic shock**
- (4) Hemorrhage
- (5) Coma.



Clinical presentation

- A sudden drop in O₂ saturation can be the initial indication of AFE during c/s.
- More than 1/2 of patients die within the first hour.
- Of the survivors 50 % will develop DIC which may manifest as persistent bleeding from incision or venipuncture sites.

The coagulopathy typically occurs 0.5 to 4 hours after phase 1.



Clinical presentation

- 10-15% of patients will develop grand mal seizures.
- CXR may be normal or show effusions, enlarged heart, or pulmonary edema.
- ECG may show a right strain pattern with ST-T changes and tachycardia.



Diagnosis

- The **presence of squamous cells in the pulmonary vasculature** once considered pathognomonic for AFE is neither sensitive nor specific (only 73% of patients dying from AFE had this finding).
- The monoclonal antibody TKAH-2 may eventually prove more useful in the rapid diagnosis of AFE.



Laboratory investigations in suspected AFE

Non specific

- complete blood count
- coagulation parameters
including FDP, fibrinogen
- arterial blood gases
- chest x-ray
- electrocardiogram
- V/Q scan
- echocardiogram

Specific

- cervical histology
- serum tryptase
- serum sialyl Tn antigen
- zinc coproporphyrin
- PMV analysis (if PA
catheter *in situ*)



Differential diagnosis

Obviously depends upon presentation

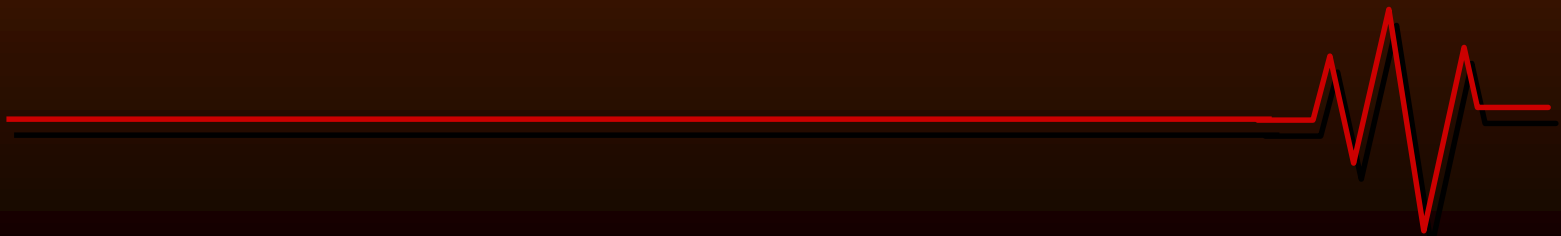
- Anaphylaxis (Collapse)
- Pulmonary embolus (Collapse)
- Aspiration (Hypoxemia)
- Pre-eclampsia or eclampsia (Fits, Coagulopathy)
- Hemorrhage (APH ; PPH)
- Septic shock
- Drug toxicity (MgSO₄, total spinal, LA toxicity)
- Aortic dissection



Management of AFE

GOALS OF MANAGEMENT:

- Restoration of cardiovascular and pulmonary equilibrium
 - Maintain systolic blood pressure >90 mm Hg.
 - Urine output > 25 ml/hr
 - Arterial pO_2 > 60 mm Hg.
- Re-establishing uterine tone
- Correct coagulation abnormalities



Management of AFE

- As intubation and CPR may be required it is necessary to have easy access to the patient, experienced help, and a resuscitation tray with intubation equipment, DC shock, and emergency medications.
- **IMMEDIATE MEASURES :**
 - Set up IV Infusion, O₂ administration.
 - Airway control ⇒ endotracheal intubation
⇒ maximal ventilation and oxygenation.
- **LABS :** CBC,ABG,PT,PTT,fibrinogen,FDP.



Management of AFE

- Treat hypotension, increase the circulating volume and cardiac output with crystalloids.
- After correction of hypotension, restrict fluid therapy to maintenance levels since ARDS follows in up to 40% to 70% of cases.
- Steroids may be indicated (recommended but no evidence as to their value)
- Dopamine infusion if patient remains hypotensive (myocardial support).
- Other investigators have used vasopressor therapy such as ephedrine or levarterenol with success (reduced systemic vascular resistance)



Management of AFE In the ICU



- To assess the effectiveness of treatment and resuscitation, it is prudent to continuously monitor ECG, pO_2 , CO_2 , and urine output.
- There is support in literature for early placement of arterial, central venous, and pulmonary artery catheters to provide critical information and guide specific therapy.



Management of AFE In the ICU

- Central venous pressure monitoring is important to diagnose right ventricular overload and guide fluid infusion and vasopressor therapy. Blood can also be sampled from the right heart for diagnostic purposes.
- Pulmonary artery and capillary wedge pressures and echocardiography are useful to guide therapy and evaluate left ventricular function and compliance.
- An arterial line is useful for repeated blood sampling and blood gases to evaluate the efficacy of resuscitation.



Management of AFE Coagulopathy

- DIC results in the depletion of fibrinogen, platelets, and coagulation factors, especially factors V, VIII, and XIII. The fibrinolytic system is activated as well.
- Most patients will have hypofibrinogenemia, abnormal PT and aPTT and low Platelet counts
- Treat coagulopathy with FFP for a prolonged aPTT, cryoprecipitate for a fibrinogen level less than 100 mg/dL, and transfuse platelets for platelet counts less than 20,000/mm³



Restoration of uterine tone

- Uterine atony is best treated with massage, uterine packing, and oxytocin or prostaglandin analogues.
- Improvement in cardiac output and uterine perfusion helps restore uterine tone.
- Extreme care should be exercised when using prostaglandin analogues in hypoxic patients, as bronchospasm may worsen the situation.



Sympathomimetic Vasopressor agent

Dopamine

- Dopamine increases myocardial contractility and systolic BP with little increase in diastolic BP. Also dilates the renal vasculature, increasing renal blood flow and GFR.
- **DOSE:** 2-5 mcg/kg/min IV; titrate to BP and cardiac output.
- **Contraindications:** ventricular fibrillation, hypovolemia, pheochromocytoma.
- **Precautions:** Monitor urine flow, cardiac output, pulmonary wedge pressure, and BP during infusion; prior to infusion, correct hypovolemia with either whole blood or plasma, as indicated; monitoring central venous pressure or left ventricular filling pressure may be helpful



Maternal Mortality in AFE

- Maternal death usually occurs in one of three ways: (1) sudden cardiac arrest, (2) hemorrhage due to coagulopathy, or (3) initial survival with death due to acute respiratory distress syndrome (ARDS) and multiple organ failure
- For women diagnosed as having AFE, mortality rates ranging from 26% to as high as 86% have been reported.
- The variance in these numbers is explained by dissimilar case definitions and possibly improvements in intensive care management of affected patients.



Further issues in the Management

- **Transfer:**
Transfer to a level 3 hospital may be required once the patient is stable.
- **Deterrence/Prevention:**
Amniotic fluid embolism is an unpredictable event.
- Risk of **recurrence** is unknown. The recommendation for elective cesarean delivery during future pregnancies in an attempt to avoid labor is controversial.
- **Perimortem cesarean delivery:**
After 5 minutes of unsuccessful CPR in arrested mothers, abdominal delivery is recommended.



Medical/Legal Pitfalls

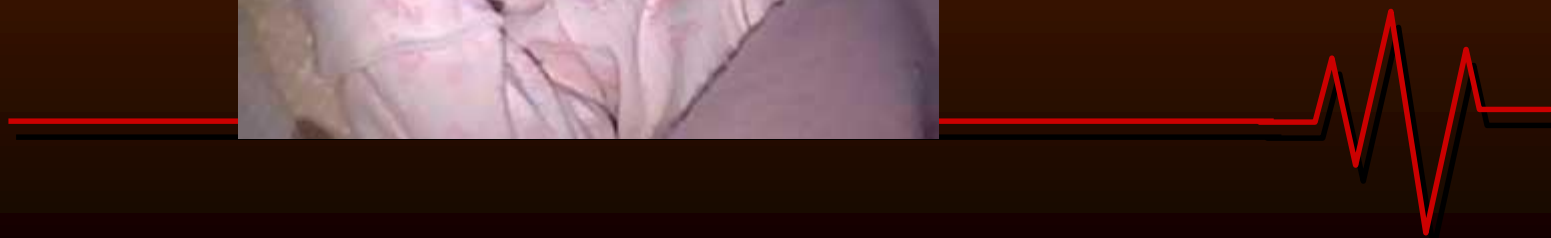
- Failure to respond emergently is a pitfall. AFE is a **clinical diagnosis**. Steps must be taken to stabilize the patient as soon as symptoms manifest.
- Failure to perform perimortem cesarean delivery in a timely fashion is a pitfall.
- Failure to consider the diagnosis during legal abortion is a pitfall. A review of the literature indicates that most case reports of AFE have occurred during late second-trimester abortions.



SUMMARY

- **AFE is a sudden and unexpected rare but life threatening complication of pregnancy.**
- **It has a complex pathogenesis and serious implications for both mother and infant**
- **Associated with high rates of mortality and morbidity.**
- **Diagnosis of exclusion.**
- **Suspect AFE when confronted with any pregnant patient who has sudden onset of respiratory distress, cardiac collapse, seizures, unexplained fetal distress, and abnormal bleeding**
- **Obstetricians should be alert to the symptoms of AFE and strive for prompt and aggressive treatment.**





Uterine Rupture

- **Uterine rupture** is a potentially catastrophic event during childbirth by which the integrity of the myometrial wall is breached.
- In an **incomplete** rupture the peritoneum is still intact. With a **complete** rupture the contents of the uterus may spill into the peritoneal cavity or the broad ligament. A uterine rupture is a life-threatening event for mother and baby.
- A uterine rupture typically occurs during early labor, but may already develop during late pregnancy



What is uterine rupture?

- The term "uterine rupture" is used for anything in a continuum of events, from a weak spot in the uterine wall noticed by the surgeon at the time of cesarean to the catastrophe of the uterus tearing open and the fetus, placenta, and a lot of blood extruding into the mother's abdomen.



Who is at risk for uterine rupture?


- Women who have had previous surgery on the uterus, particularly on the upper muscular portion
 - 1. cesareans that were *not* low transverse are at increased risk for uterine rupture. Prior classical cesareans
 - 2. *multiple* (three or more) prior low transverse cesareans all put a pregnant woman at increased risk.
 - 3. prior **removal of fibroid tumors** if the incision extended through the full thickness of the uterine wall
 - 4. any other **uterine surgery** that went through the full depth of the muscular portion of the uterus



Who is at risk for uterine rupture?

- Even without prior surgery, having had more than five full-term pregnancies, having an overdistended uterus (as with twins or other multiples)
- abnormal positions of the baby such as transverse lie, macrosomia.
- the use of Pitocin and other labor-inducing medications like prostaglandins may increase the risk.

There is no evidence that D&C, first-trimester abortion, removal of superficial fibroids, or pelvic surgery that did not involve the uterus increase the risk.



What is the risk?

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Mild type uterine ruptures occur without symptoms and do not cause problems for the mother or fetus. only noticed when surgery.

- severe form of uterine rupture, where the laceration is large or cuts across the uterine blood vessels, the mother may hemorrhage and require a blood transfusion, the uterus may not be repairable and must be surgically removed (hysterectomy), the baby may not survive the lack of oxygen, and (rarely) the mother's life, too, cannot be saved.

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What is the risk?

- The uterus can rupture before or during labor.
- mothers who had one previous low transverse cesarean, the risk of uterine rupture :
 - 1 per 625 women who chose repeat cesarean without labor
 - 1 per 192 women who went into labor and tried for VBAC
 - 1 per 129 for those who had their labor induced without prostaglandins (usually with Pitocin),
 - 1 per 41 when **prostaglandin medications** were used for induction.
- When the uterus did rupture:
 - 1 in 18 babies died
 - 1 in 23 of the women required a hysterectomy.



Symptoms and Signs of uterine rupture

- largely depend on the timing, site, and extent of the uterine defect.
 - Uterine rupture at the site of a previous uterine scar is typically less violent and less dramatic than a spontaneous or traumatic rupture because the scar is relatively avascular.
1. Abdominal pain and tenderness.

The pain may not be severe; it may occur suddenly at the peak of a contraction.
 2. Chest Pain, pain between the scapulae, or pain on inspiration—



3.Hypovolemic Shock :

caused by hemorrhage- Falling blood pressure, tachycardia, tachypnea, cool and clammy skin, and anxiety. The fall in blood pressure is often a late sign of hemorrhage

4.Signs associated with fetal oxygenation:

late decelerations, reduced viability, tachycardia, and bradycardia

Absent fetal heart sounds with a large disruption of the placenta;
absent fetal heart activity by ultrasound examination



Preventing uterine rupture

- Some uterine ruptures occur before labor and are considered unpreventable.
- Sudden severe abdominal pain in later pregnancy should be reported to your physician, especially if you are at increased risk for rupture of the uterus.
- Women with risk factors should not attempt labor, and should be scheduled for cesarean as soon as the fetus is expected to do well out in the world, usually between 36 and 39 weeks' gestation.



Preventing uterine rupture

- For women at some increased risk of rupture, fetal monitoring during labor can alert our doctors that this complication is developing. Labor after cesarean should be undertaken only in hospitals where emergency surgery is available.
- it became clear that after prior cesarean, the greatest risk of uterine rupture occurs when labor is induced using prostaglandin medications.

